UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
PREVENTION, PESTICIDES
AND TOXIC SUBSTANCES

Note to Reader

Background: As part of its effort to involve the public in the implementation of the Food Quality Protection Act of 1996 (FQPA), which is designed to ensure that the United States continues to have the safest and most abundant food supply. EPA is undertaking an effort to open public dockets on the organophosphate pesticides. These dockets will make available to all interested parties documents that were developed as part of the U.S. Environmental Protection Agency's process for making reregistration eligibility decisions and tolerance reassessments consistent with FQPA. The dockets include preliminary health assessments and, where available, ecological risk assessments conducted by EPA, rebuttals or corrections to the risk assessments submitted by chemical registrants, and the Agency's response to the registrants' submissions.

The analyses contained in this docket are preliminary in nature and represent the information available to EPA at the time they were prepared. Additional information may have been submitted to EPA which has not yet been incorporated into these analyses, and registrants or others may be developing relevant information. It's common and appropriate that new information and analyses will be used to revise and refine the evaluations contained in these dockets to make them more comprehensive and realistic. The Agency cautions against premature conclusions based on these preliminary assessments and against any use of information contained in these documents out of their full context. Throughout this process, If unacceptable risks are identified, EPA will act to reduce or eliminate the risks.

There is a 60 day comment period in which the public and all interested parties are invited to submit comments on the information in this docket. Comments should directly relate to this organophosphate and to the information and issues available in the information docket. Once the comment period closes, EPA will review all comments and revise the risk assessments, as necessary.

These preliminary risk assessments represent an early stage in the process by which EPA is evaluating the regulatory requirements applicable to existing pesticides. Through this opportunity for notice and comment, the Agency hopes to advance the openness and scientific soundness underpinning its decisions. This process is designed to assure that America continues to enjoy the safest and most abundant food supply. Through implementation of EPA's tolerance reassessment program under the Food Quality Protection Act, the food supply will become even safer. Leading health experts recommend that all people eat a wide variety of foods, including at least five servings of fruits and vegetables a day.

Note: This sheet is provided to help the reader understand how refined and developed the pesticide file is as of the date prepared, what if any changes have occurred recently, and what new information, if any, is expected to be included in the analysis before decisions are made. **It is not meant to be a summary of all current information regarding the chemical.** Rather, the sheet provides some context to better understand the substantive material in the docket (RED chapters, registrant rebuttals, Agency responses to rebuttals, etc.) for this pesticide.

Further, in some cases, differences may be noted between the RED chapters and the Agency's comprehensive reports on the hazard identification information and safety factors for all organophosphates. In these cases, information in the comprehensive reports is the most current and will, barring the submission of more data that the Agency finds useful, be used in the risk assessments.

Jack E. Housenger, Acting Director
Special Review and Reregistration Division

Portions of this document (Pages 16 - end) have been claimed confidential. This document is releasable to persons who submit a signed "Affirmation of Non-Multinational Status" form.

DDVP-cancer

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

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March 7, 2000

MEMORANDUM

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

SUBJECT: Diclorvos (DDVP) - Report of the Cancer Assessment Review Committee

FROM: Sanjivani Diwan, Executive Secretary Sanjivani Diwan
Cancer Assessment Review Committee
Health Effects Division (7509C)

TO: Joycelyn Stewart, Pharmacologist
Science Analysis Branch
Health Effects Division (7509C)

David Jaquith, Risk Assessor
Reregistration Branch 4
Health Effects Division (7509C)

Kimberly Lowe, Chemical Review Manager
Special Review Branch
Special Review and Reregistration Division (7505C)

The 6th Cancer Assessment Review Committee met on August 18, 1999 to evaluate the carcinogenic potential of Diclorvous (DDVP). Attached please find the Final Cancer Assessment Document.

cc: L. Brunsman
W. Burnam
M. Copley
K. Dearfield
V. Dobozy
R. Hill
Y. Ioannou
N. McCarroll
T. McMahon
J. Pletcher
E. Rinde
J. Rowland
J. Stewart
C. Swentzel
L. Taylor

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

014029

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

February 2, 2000

MEMORANDUM

Subject: *Diclorvos*-CARC Final Report

From: William Burnam
Chairman,
Cancer Assessment Review Committee

To: CARC Members

I am circulating my revised write-up on the carcinogenic potential of DDVP based on discussions during the sixth CARC meeting held last August, 1999. I am looking for a consensus and believe that my summary reflects that. If enough CARC members favorably sign the document, I will assume that this represents a consensus opinion. If you think that this report does not reflect your opinions, please indicate this also. If there is no clear consensus, we will meet again on DDVP soon.

HED DOC. NO. 014029

CANCER ASSESSMENT DOCUMENT

EVALUATION OF THE CARCINOGENIC POTENTIAL OF

DICLORVOS *(DDVP)*

(SIXTH REVIEW)

FINAL REPORT

1-MARCH-2000

CANCER ASSESSMENT REVIEW COMMITTEE
HEALTH EFFECTS DIVISION
OFFICE OF PESTICIDE PROGRAMS

DATA PRESENTATION: _____
Joycelyn Stewart, Toxicologist

DOCUMENT PREPARATION: _____
William Burnam, Chairman

COMMITTEE MEMBERS IN ATTENDANCE: (Signature indicates concurrence with the assessment unless otherwise stated).

Karl Baetcke _____

Kerry Dearfield _____

Vicki Dellarco _____

Sanjivani Diwan (Exe. Secretary) _____

Virginia Dobozy _____

Richard Hill _____

Yiannakis Ioannou _____

Nancy McCarroll _____

Esther Rinde _____

Jess Rowland _____

Clark Swentzel _____

Linda Taylor _____

NON-COMMITTEE MEMBERS IN ATTENDANCE (Signature indicates concurrence with the pathology report and statistical analysis of data, respectively)

John Pletcher, Pathology _____

Lori Brunsman, Statistical Analysis _____

CONTENTS

SUMMARY

At the 6th Cancer Assessment Review Committee (CARC) meeting for DDVP held on August 18, 1999, the Committee determined that DDVP should remain classified as a category C carcinogen with low dose risk extrapolation based on the incidence of forestomach tumor (squamous cell papilloma and/or carcinoma) in female mice. Previously, DDVP had been classified as a category C, but the low dose linear extrapolation had been based on mononuclear cell leukemia (MCL) in male rats.

After the August 18 meeting, an E-mail message was sent out expressing concern that the DDVP classification was not based on the Agency's new draft Cancer Risk Assessment Guidelines (1999). In addition, the CARC had previously (in its 5th Peer Review) decided not to use the forestomach tumors for linear extrapolation. The members of the CARC were requested to consider changing the conclusions of the August 18, 1999, meeting on DDVP. The CARC member's opinions were at least two to one in favor of changing the classification to "suggestive" and not requiring a low-dose linear extrapolation. The following is a rationale to support this conclusion.

At the 5th Cancer Peer Review of DDVP (report dated August 2, 1996), the conclusion was that the new information on MCL staging did not negate the Committee's previous conclusion that this tumor was treatment related. The mouse forestomach tumors were still considered caused by DDVP but, since their relevance to human health was in doubt, they were not included in the linear low dose extrapolation. The Committee concluded that the classification should remain a Category C based on these two tumor types with a linear low dose risk assessment based on the MCL.

The discussion at the 6th CARC meeting centered around the significance of the MCL in the male rats and their relevance for risk assessment based on three separate papers sent to the CARC prior to the meeting (1) the registrant's July 27, 1998, "An Evaluation of the Potential Carcinogenicity of Dichlorvos: Final Report of the Expert Panel"; (2) the report of the FIFRA SAP meeting of July 30, 1998; and (3) a memorandum of a phone conversation between Dr.Boorman of NTP and certain CARC members. A few of the reasons are as follows:

1) MCL is common in the Fischer rat and, in the males, appears to vary in its background rate with the amount of corn oil in the animal's diet.

2) The tumor type does seem to be found mainly in this Fischer strain and does not appear to be similar to leukemia in humans (adults or children).

3) There was no dose response in the incidence and severity between the two gavage doses of 4 and 8 mg/kg/day.

The overall conclusion of CARC was that, while all of this information somewhat lessened our concern, the MCL could not be totally dismissed as not being relevant to humans. This agreed with the opinion of Dr. Boorman of NTP.

The CARC also agreed in principle, with the SAP's statement "overall, the high background and variability in the incidence of this tumor, as well as its species and strain specificity, make it an invalid response for human risk assessment." Based on these conclusions and, after an informal poll of the CARC, it was determined that "suggestive" under the 1999 Draft Agency Cancer Guidelines best described the carcinogenic potential of DDVP. The rationale can be stated as follows:

1) MCL in the male Fischer rat has certain properties in terms of variability and reliability which limit its usefulness for human risk assessment.

2) The forestomach tumors, observed at gavage doses causing inhibition of plasma and red blood cell cholinesterase and cholinergic signs, are also limited in their use for human risk assessment.

3) The fact that DDVP is only positive by the gavage route and negative by the inhalation route,which is the major route of human exposure, indicates that any classification by the oral route may be limited since localized effects in the forestomach may not be applicable to human risk assessment.

Attachments

ATTACHMENT 2

FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT

SCIENTIFIC ADVISORY PANEL MEETING

A Set of Scientific Issues Being Considered by the Agency in Connection with DDVP (Dichlorvos) Risk Issues

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The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Scientific Advisory Panel (SAP) has completed its review of the set of scientific issues being considered by the Agency in connection with DDVP (Dichlorvos) Risk Issues. The review was conducted in an open meeting held in Arlington, Virginia, on July 30, 1998. The meeting was chaired by Dr. Ernest E. McConnell (ToxPath, Inc.). Other Panel Members present were: Dr. Janice Chambers (Mississippi State University); Dr. Rory Conolly (Chemical Industry Institute of Toxicology-CIIT); Dr. Michael Cunningham (National Institute of Environmental Health Sciences-NIEHS); Dr. Amira Eldefrawi (University of Maryland School of Medicine); Dr. Richard Fenske (University of Washington); Dr. David Gaylor (National Center for Toxicological Research); Dr. Charles Hobbs (Lovelace Respiratory Research Institute); Dr. Gordon Hard (American Health Foundation); Dr. Ronald J. Kendall (The Institute of Environmental and Human Health, Texas Tech University/Texas Tech University Health Sciences Center); Dr. Ross Leidy (North Carolina State University); Dr. Genevieve M. Matanoski (The Johns Hopkins University); Dr. Fumio Matsumura (University of California); Dr. Herbert Needleman (University of Pittsburgh); Dr. Christopher Portier (National Institute of Environmental Health Sciences-NIEHS); Dr. J. Routt Reigart (Medical University of South Carolina); Dr. Mary Anna Thrall (Colorado State University); and Dr. John Wargo (Yale University).

Public Notice of the meeting was published in the Federal Register on June 19, 1998.

Oral statements were received from the following:

Robert Becker, MD, representing SRA International, Inc.

Dr. Bruce Bernard (SRA International, Inc.)

Dr. David Brusick (Covance)

Dr. Samuel Cohen (University of Nebraska), representing SRA International, Inc.

Dr. Jay Goodman (Michigan State University), representing SRA International, Inc.

Dr. Stephen Harris (Stephen B. Harris Group)

Mr. Bill Harvack (Amvac Corporation)

Dr. Judy MacGregor (Toxicology Consulting Services)

Dr. Rudy Richardson (University of Michigan), representing SRA International, Inc.

Dr. Jay Schreider (California Department of Pesticide Regulation)

Dr. David Wallinga (National Resources Defense Council)

Dr. Susan Youngen (Novigen Sciences)

Written statements were received from the following:

SRA International

General Comments from SAP Members

The Panel reviewed a considerable amount of material related to Dichlorvos (DDVP), including the PD 2/3 (Special Review Action, Notice of Intent to Cancel) issued by the Agency in September, 1995. Agency response to public comments received on the PD 2/3 was completed in June, 1996. In the PD2/3, the Agency proposed cancellation of DDVP uses for: (1) residences (including pet flea collars, total release foggers, pressurized aerosols, crack and crevice treatment); (2) tobacco warehouses; (3) ornamental lawns; (4) turf and plants; (5) commercial, institutional and industrial areas; (6) commercial vehicles (airplanes, trucks, shipholds and rail cars); (7) warehouses; (8) bulk, packaged, or bagged nonperishable processed and raw foods and; (9) hand-held application in mushroom houses, greenhouses and passenger buses.

The July 8, 1998, DDVP Risk Assessment Issues paper prepared by the Agency stated that since these events, the registrant had voluntarily cancelled uses in tobacco warehouses and in commercial transportation vehicles. The Panel inquired as to what actions, if any, the Agency has taken in regard to the other uses for which cancellation was proposed. It appears that the Agency has continued its analysis of toxicity and exposure data for DDVP during this time, but has not proceeded with any regulatory actions. It was not clear that such reanalyses have changed significantly the risk assessments which underlay the PD 2/3 document. The Panel urged the Agency to clarify remaining risk assessment issues for DDVP and to move forward, where appropriate, with proposed regulatory actions.

The Panel recognized the substantial number of public comments at this session. Scientists representing the registrant presented the results of an independent advisory panel's deliberations on the genotoxicity and carcinogenicity of DDVP, as well as the use of cholinesterase inhibition data in the regulatory process. These presentations in turn raised questions regarding neurotoxicity testing for DDVP. The Panel is providing the following comments and concerns on these topics.

Genotoxicity Comments:

The Panel noted that the registrant has submitted data from a series of mechanistic studies in an attempt to demonstrate that dichlorvos is a nongenotoxic agent similar to butylated hydroxyanisole (BHA) and not a genotoxic agent like 1-methyl-3-nitro-1-nitrosoguanidine

(MNNG). The studies attempted to prove that dichlorvos induces replicative DNA synthesis (RDS) and cell proliferation and does not induce unscheduled DNA synthesis (UDS) indicative of DNA damage. The experimental design of these studies is unvalidated and flawed. Mice were administered a single dose of dichlorvos, BHA or MNNG or vehicle control by gavage. Animals were serially sacrificed up to 48 hours later. Forestomachs were removed for histopathological examination or incubated with 3H-thymidine in vitro for assessment of UDS or RDS. These data cannot be evaluated for several reasons. Single dose studies have little relationship to the longer term toxicity of a chemical. Acute effects seldom predict chronic effects. In the present experimental design, any UDS in the forestomach induced by dichlorvos would be masked by the RDS, since the RDS measurement evaluates an entire nucleus darkened by 3H-thymidine and UDS evaluates small dots of radioactivity incorporated into the nucleus. Simultaneous measurements are not compatible. Additionally, *in vitro* administration of the label is not reliable and has not been validated as has *in vivo* administration of the label.

A parallel study measured 3H-thymidine incorporation/mg DNA. This is an outdated technique that provides meaningless data without information about the subcellular location of the label or the cell types involved.

Overall, the UDS assay has been shown to be quite insensitive to mutagenic chemicals with the exception of potent alkylating agents. A positive response is a clear indication of DNA damage, but a negative does not indicate nongenotoxicity.

The Panel noted that mutagenic batteries were developed in order to provide a rapid and inexpensive screen to determine the potential of a chemical to induce cancer. They are generally simple tests with a narrow response. These data from these tests are generally reproducible in that a chemical positive in one test is generally positive in other similar tests. There is a fairly poor concordance (both positive and negative predictivity) for the results of chronic bioassays such as those conducted by the NIH National Toxicology Program (NTP). The best test to date is the Ames test, which has about 65 percent concordance. This includes almost 20 percent of chemicals judged positive in the Ames test that result in negative rodent cancer results. These "false positives" (or genotoxic noncarcinogens) result not from a failure of the *in vitro* test but from the complexity of the carcinogenesis process in the whole animal. These processes in the intact animal that are not reflected in mutation assays include absorption, disposition, metabolism and elimination (ADME) considerations, cytotoxicity resulting in compensatory hyperplasia, mitogenesis, metabolic enzyme induction or inhibition, etc.

Therefore, the results from whole animal bioassays supersede the results of *in vitro* tests. Compounds that are inactive in mutation tests but cause cancer in the whole animal are considered nonmutagenic and carcinogenic and generally are regulated as carcinogens in the U.S. Similarly, compounds that are positive in mutation tests but do not cause cancer in the whole animal should be regulated as noncarcinogens.

Carcinogenicity Comments:

Several Panel members commented on the carcinogenicity of dichlorvos. There is an emerging view based on cumulative experience by some toxicologic pathologists that mononuclear cell leukemia in the Fischer rat may be a unique type of cancer and not induced *de novo* by compound administration. In the case of DDVP, both low and high doses showed a doubling of mononuclear cell leukemia (MCL) incidence without a clear dose-response relationship and only a tenuous link with respect to carcinogenic potential. Concerning the mouse forestomach tumors, DDVP joins a group of compounds that can cause irritation and/or breakdown of the physiological gastric mucosal barrier and, indirectly, forestomach tumors through sustained cytotoxicity and cell regeneration. Thus, quantification of the cancer risk assessment of this compound should be a nonlinear threshold approach based on the forestomach tumors. The negative cancer ingestion bioassay in rats contributes to the weight-of-evidence evaluation of the carcinogenic potential of DDVP. In particular, this study reduces the likelihood that the rat leukemia seen in the corn oil gavage study is due to a directly genotoxic mechanism of DDVP. Such an effect is not expected to show a route-of-administration effect.

inhalation

There is compelling evidence to disregard MCL in the Fischer rat. MCL is one of the most common background tumor types in this strain, and has been referred to as Fischer rat leukemia. Other rat strains and mice do not develop MCL, and there is no human correlate to this disease. Additionally, chemically-related increases in MCL exhibit advanced severity grades for this lesion in treated rats compared to controls. Analysis of the MCL in the DDVP gavage study showed no significant increase in severity of the MCL with increasing dose, indicating that these lesions are background. Overall, the high background and variability in the incidence of this tumor, as well as its species and strain specificity, make it an invalid response for human risk assessment. However, it should be noted as in the case of benzene carcinogenicity, early studies showed carcinogenicity in Zymbal's gland in rats for which there is also no human correlate.

Forestomach carcinogenesis in oral gavage bioassays in the absence of glandular stomach or any other target organ carcinogenesis is likely due to the chronic irritancy, inflammation, and cytotoxicity during chronic bolus dosing, resulting in extraordinarily high local concentration of the chemical. This chronic toxicity to the forestomach puts cells into chronic mitosis and provides an environment for heightened sensitivity to food-borne carcinogens endogenous in diets or to the likelihood of promotion of naturally occurring, spontaneous background tumor cells being stimulated to divide. In addition, the forestomach in rodents acts as a storage site where irritant chemicals in the food have prolonged contact with the sensitive squamous epithelium lining, a situation that does not pertain to humans. Thus, the feeding study of DDVP, which did not induce tumors in the forestomach or any organ, should be taken into consideration.

One Panel member noted that in assessing the overall carcinogenicity of DDVP, the Agency should be careful of overly simplistic interpretation of p-values. The fact that the leukemia p-value for the female rats does not achieve statistical significance is not of as great an interest as the fact that the marginal finding ($p=0.07$) in females supports the significant finding in males. In addition, the marginal finding in the Japanese study is also in the same direction as that seen in the males from the NTP study, but for a different route for which absorption could be remarkably different.

In conclusion, the weight of the evidence suggests carcinogenicity in animals treated with DDVP with a non-linear dose-response. However, the compound is considered a weak carcinogen acting via a secondary or indirect mechanism.

Cholinesterase Inhibition Comments:

The Panel noted that because of the vital role played by acetylcholine (ACh), acetylcholinesterase (AChE) exists in several-fold excess at cholinergic synapses and a great deal more in the blood, presumably to protect the body against this highly bioactive molecule. The various cholinesterase isozymes differ in their response to organophosphates. Plasma cholinesterase is usually more sensitive than red blood cell acetylcholinesterase. The least sensitive is usually AChE, which is located in cholinergic synapses (in muscles, glands, and brain), and in some cases the degree of its inhibition correlates best with toxicity symptoms. The blood enzymes act as reservoirs for removing organophosphates from circulation, thus reducing their concentrations at the critical targets. These are the cardiac, smooth and skeletal neuromuscular and neuroglandular acetylcholinesterases. However, Panel members differed on whether inhibition at these targets correlates best with the toxicity symptoms. A Panel member commented that there are limited data on inhibition of peripheral cholinesterase.

No-observable-adverse-effect-level (NOAEL) or lowest-observable-adverse-effect-level (LOAEL) values, based on inhibition of plasma or blood cholinesterases, do not necessarily reflect toxicity and may in some cases give higher values, which is in fact less conservative. In terms of acute studies, Panel members provided different opinions on the best approach for measurement of acetylcholinesterase inhibition. One Panel member commented that the best approach to measure inhibition of acetylcholinesterase is in cardiac, skeletal, smooth muscles or brain. However, another Panel member remarked that the methodology is problematic for measuring acetylcholinesterase inhibition in cardiac, skeletal, or smooth muscles. Thus, consideration of acetylcholinesterase inhibition in cardiac, skeletal, or smooth muscles would be premature at this time. In terms of subchronic and chronic studies, development of tolerance should also be considered. While data support the idea that high levels of brain cholinesterase inhibition are required to elicit clinical signs of toxicity, 60 percent brain cholinesterase inhibition is too great to consider as a threshold for adverse effects.

It is important for the Agency to adopt consistent use of the terms NOEL and NOAEL with its documents and decisions.

Neurotoxicity Testing Comments:

In the discussion of cholinesterase inhibition, the Panel raised concerns regarding the suggestion that clinical signs should serve as an indication of adverse effects compared to more subtle behavioral modifications, which should be considered appropriate for judging toxic responses. In 1996, the SAP recommended that the Agency develop validated test protocols for Scheduled Analysis of Operant Behavior (SCOB) studies to be used to test neurotoxins. A guideline for the SCOB is available. This type of testing, or any behavioral data used to determine the toxicant's neurotoxicity, has not been conducted for DDVP.

DICHLORVOS
(Group 2B)

For definition of Groups, see Preamble Evaluation.

VOL.: 53 (1991) (p. 267)

CAS No.: 62-73-7
Chem. Abstr. Name: Phosphoric acid, 2,2-Dichloroethenyl dimethyl ester

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Dichlorvos has been used widely as an insecticide since 1961 to control internal and external parasites in livestock and domestic animals, to control insects in houses, and in crop protection.

Dichlorvos has been formulated for use as dusts, granules, pellets/tablets, impregnated resin strips and concentrates.

Household and public health uses represent the main sources of human exposure to dichlorvos. Exposure may also occur during its production and application.

5.2 Carcinogenicity in humans

One case-control study of leukaemia in the USA found an association with use of dichlorvos on animals; there were few exposed subjects, and they had potential exposure to many pesticides.

5.3 Carcinogenicity in experimental animals

Dichlorvos was tested for carcinogenicity by oral administration in two experiments in mice and in three experiments in rats. A few rare oesophageal squamous-cell tumours were found in mice treated with dichlorvos in the diet. A dose-related increase in the incidence of squamous-cell tumours (mainly papillomas) was noted in the forestomachs of mice that received dichlorvos in corn oil by gavage. In rats that received dichlorvos in water by gavage, a few squamous-cell papillomas of the forestomach were seen. In rats that received dichlorvos in corn oil by gavage, a dose-related increase in the incidence of mononuclear-cell leukaemia and an increased incidence of pancreatic acinar-cell adenomas were observed in males.

5.4 Other relevant data

A variety of studies in several species did not demonstrate developmental toxicity due to dichlorvos.

In vitro, dichlorvos phosphorylates esterases to a greater extent than it methylates nucleophiles; the likelihood of DNA methylation *in vivo* is extremely small.

Immunosuppression has been noted after short-term administration of high doses of dichlorvos which are associated with profound cholinergic hyperstimulation.

No data were available on the genetic and related effects of dichlorvos in humans.

Dichlorvos was not shown to have genetic activity in various assays in mammals in vivo. It induced gene mutation and chromosomal damage in cultured mammalian cells and in insects, plants, fungi, yeast and bacteria.

5.5 Evaluation

There is *inadequate evidence* in humans for the carcinogenicity of dichlorvos.

There is *sufficient evidence* in experimental animals for the carcinogenicity of dichlorvos.

Overall evaluation

Dichlorvos is *possibly carcinogenic to humans (Group 2B).*

For definition of the italicized terms, see Preamble Evaluation.

Previous evaluations: Vol. 20 (1979) (p. 97); Suppl. 7 (1987) (p. 62)

Synonyms for Dichlorvos

- Atgard
- Atgard V
- Bibesol
- Brevinyl
- Brevinyl E50
- Canogard
- Chlorvinphos
- DDVP
- Dedevap
- Des
- Dichlofos
- Dichlorman
- 2,2-Dichloroethenyl dimethyl phosphate
- 2,2-Dichloroethenol, dimethyl phosphate
- Dichlorovos
- Dimethyl 2,2-dichloroethenyl phosphate
- Dimethyl dichlorovinyl phosphate
- Dimethyl 2,2-dichlorovinyl phosphate
- *O,O*-Dimethyl 2,2-dichlorovinyl phosphate
- Divipan
- ENT 20738
- Equigard
- Equigel
- Estrosel
- Estrosol
- Fecama
- Fekama
- Herkol
- Insectigas D
- Mafu Strip
- Mopari
- Nefrafos
- Nerkol
- Nogos
- Nogos 50
- Nogos G
- Novotox
- No-Pest Strip
- Nuan
- Nuvan
- Nuvan 100 EC
- OKO